Post-Traumatic Parotid Fistulae and Sialoceles

A Prospective Study of Conservative Management in 51 Cases

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The management of parotid sialoceles and fistulae have been unsatisfactory in the past, and numerous methods of treatment with varying success and morbidity have been described. The present prospective study reports results of conservative therapy in 51 patients over a 3-year period. In 50 patients, the injury healed upon conservative management. During the early phase of the study, a limited conservative regimen through which the patients received nothing orally for 5 days only was used. During the latter part of the study, patients were administered nothing orally until complete healing of the injury. In terms of the time it took for healing of the injury, the differences of the two regimens (24 ± 4 *vs.* 9.4 ± 0.9 days) was highly significant ($p < 0.001$). The response to conservative management depended on the severity of injury as demonstrated by sialography. Injury to minor intraparotid ducts (G1) healed in significantly less time compared with that to a major intraparotid duct (G2) or ductal injuries ($p < 0.001$). There was no difference between the healing of G2 injury (10.3 ± 1.8 days) and partial ductal transections (10.5 ± 2.2 days) ($p > 0.05$). There was a significantly greater delay in healing with complete duct transections (21.5 ± 3.7 days) compared with partial duct transections and G2 injuries (10.2 ± 2.1 days) ($p < 0.01$). There was no difference in the mean period for healing between salivary fistulae and sialoceles ($p > 0.05$). It is concluded that a new classification of parotid fistulae based on sialographic findings has prognostic and therapeutic value. Furthermore, the excellent results achieved with conservative therapy in this study suggest that it may be the initial treatment of choice for parotid fistulae.

THE MAJOR CAUSES of parotid trauma in a civilian practice are penetrating injury to the parotid gland from an assault weapon or from injury due to shattered glass after a motor vehicle accident (MVA). Patients whose initial injury is missed on admission may present at varying intervals with a parotid fistula that may be due either to a glandular or a ductal injury. An internal parotid fistula commonly presents as a sialocele or an effusion, depending on when the diagnosis is made, on when the diagnosis is made, whereas communication with the skin leads to an external parotid fistula.

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Although there is consensus in the literature that acute parotid injury must be explored primarily and all injured structures be repaired accurately,[1] the treatment of the missed parotid injury is controversial. Numerous methods of treatment (Tables 1 and 2)[2–28] have been described with varying success and morbidity. We describe results of conservative management in 51 cases of parotid fistulae and sialoceles. Preliminary results from a pilot study have been reported previously.[29]

Patients and Methods

All patients with a parotid duct or gland injury from trauma that was diagnosed more than 72 hours after the injury were entered into this study for conservative management during the period of 1985–1987. Patients whose injury presented early were treated with primary repair of all damaged structures. All patients had a sialogram before commencement of conservative treatment to document the site of injury. In some patients a radioisotope dynamic flow scan with radioactive technesium pertechnetate was performed to assess the function of the gland. Patients were administered nothing orally, with provision of maintenance fluids administered intravenously. During the pilot stage of the study, the patients were administered nothing orally for only 5 days with Pro-banthine and pressure bandage (Regimen 1). However, due to the prolonged period required for healing of the injury with this regimen, the patients were administered nothing orally until complete healing of the injury (Regimen 2) during the latter part of the study. The results of the two regimens are compared in this study. We also abandoned the use of anti-sialogogues, pressure bandages, and repeated as-

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TABLE 1. *Management of Parotid Sialoceles and Fistulae: A Classification of Reported Methods in the Literature*

1. Diversion of parotid secretion into the mouth
   A. Reconstructive methods
      Delayed primary repair of duct
      Reconstruction of duct with vein graft
      Mucosal flaps
      Suture of proximal duct to buccal mucosa
   B. Formation of a controlled internal fistula
      T-tube or catheter drainage into the mouth
      Drainage of proximal duct by a catheter
   C. Parotidectomy
   D. Local therapy to the fistula
      Excision
      Cauterization
2. Depression of parotid secretion
   A. Surgical approaches
      Duct ligation
      Sectioning of the auricotemporal or Jacobsen's nerve
   B. Conservative approaches
      Administering nothing orally to the patient until the fistula closes
      Drugs: atropine or Pro-banthine
      Radiotherapy
      Repeated aspiration and pressure dressing

pirations. Patients with fistulae that persisted beyond 8 days were provided with parenteral nutrition (2000 kcal daily) by the peripheral route.[30] The sialogram was assessed by an independent observer who was not aware of the clinical course of the patient.

There were 45 men and six women in this study. The mean age was 28 years (range of 16–57 years). These figures clearly reflect the population of young males who

TABLE 2. *Management of Parotid Fistulae and Sialoceles Reported in the Recent Literature (1960–1987)*[2-28]

	Glandular Injury		Ductal Injury	
	No.	Recurrent Disease	No.	Recurrent Disease
Probanthine/aspiration/ pressure bandage	7	0	2	0
Controlled internal drainage	1	0	11	0
Duct ligation	0	0	9	0
Radiotherapy	7	2	5	3
Parotidectomy	0	0	11	1
Tympanic neurectomy*	6	0	2	2
Duct repair	0	0	9	5
Saphenous vein graft	0	0	1	0
Wire seton	0	0	1	0
Marsupialization	1	0	0	0
No treatment	8	0	0	0
Total number of patients		28		40
Total number procedures		30		52

* An additional 20 procedures are reported where the type of injury is not specified.[27] The mean healing period for fistulae in this study was 3.6 months (3–5 months).

TABLE 3. *Time to Presentation and to Healing for Parotid Injury*

	No.	Time to Present	Time to Healing
Fistula	15	7	14.6 ± 3.0
Sialocele	28	12	16.3 ± 3.0
Effusion	8	1.5	10.1 ± 1.5
		$p < 0.001$	$p > 0.05$

are at risk for civilian trauma. All the injuries were sustained by penetrating injury from an assault weapon or shattered glass after an MVA. In all patients, the injury was missed at the initial presentation. Ten patients had additional injuries to the chest (six patients with hemothorax) and the head (four patients with fractured skulls), and in three patients, the parotid injury was associated with fracture of the mandible. Nineteen patients (37%) had associated facial nerve injury mainly affecting the buccal and zygomatic branches.

Statistical Analysis

The results are expressed as mean ± SEM. The differences between the groups were analyzed using the Kruskal Wallis test. A Bonferroni correction was used where appropriate for one-to-one comparison within groups.

Results

The parotid injury presented clinically as sialocele in 28 patients (58%), fistula in 15 (30%), and effusion in eight (15%) (Table 3). The patients with a parotid effusion presented with a soft tissue swelling that was misdiagnosed as hematoma or soft tissue injury, and parotid injury was diagnosed after a mean of 4.2 days in the eight patients. The delay in presentation with a sialocele was significantly greater compared with an effusion or a fistulae ($p < 0.01$) (Table 3). Infective complications occurred in 13 patients (26%). Two patients had superficial wound sepsis that resolved rapidly with local dressings. In eleven patients, infection complicated a sialocele. In 73% (eight of eleven patients) of the patients of this group, the sialocele was converted to an external salivary fistula communicating with the skin. In all eleven patients, antibiotic therapy with ampicillin led to rapid resolution of the sepsis, with only two patients requiring an incision and drainage for an infected sialocele.

There were no complications associated with sialography in the 51 patients. The response to conservative therapy depended on the nature of the injury documented on the sialogram. Based on this experience, we propose a new classification for parotid sialoceles and fistulae that has a therapeutic significance (Table 4) (Figs. 1–4). The injury healed in 50 patients (Table 5). The single patient in whom there was a persistent sialocele was lost to follow-

TABLE 4. *A New Classification of Parotid Injury (Based on Sialographic Appearances)*

Glandular Injury
Type 1: Injury to the parenchyma or to minor ducts (G1)
Type 2: Injury to a major intraparotid duct (G2)
Ductal Injury
Type 1(a): Partial transection of the parotid duct [D1(a)]
Type 1(b): Complete transection of parotid duct [D1(b)]
Type 2(a) Partial disruption of parotid gland-duct junction [D2(a)]
Type 2(b) Complete disruption of parotid gland-duct junction [D2(b)]

up after only 5 days of conservative therapy (Regimen 1). There were highly significant differences in the healing period between the different types of injuries ($p < 0.005$). G1 injury healed in significantly less time (6.3 ± 0.07 days) compared with G2 and ductal injuries ($p < 0.001$). There was no difference between the healing period of G2 injury (10.3 ± 1.8 days) and partial duct [D1(a), D2(a)] injuries (10.1 ± 2.2 days) ($p > 0.05$). There was significantly greater delay in healing with complete ductal transection (21.5 ± 3.7 days) compared with partial ductal transection and G2 injury (10.2 ± 2.1 days) ($p < 0.01$).

There was no difference in the healing period with the use of Pro-banthine or pressure dressings ($p > 0.05$). There was highly significant delay in healing with Regimen 1 (24 ± 4 days) compared with Regimen 2 (9.4 ± 0.9 days)

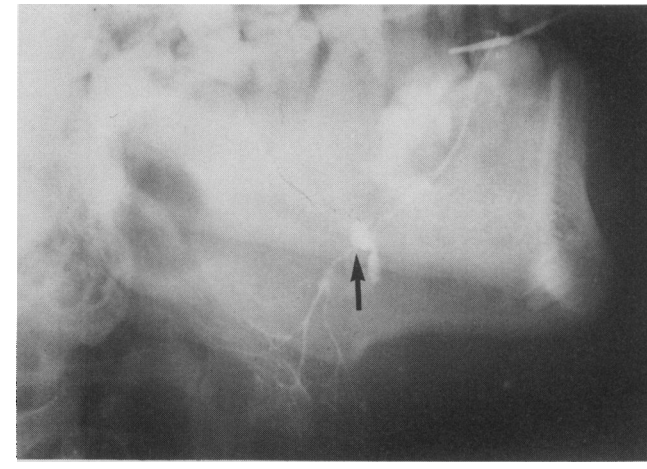

FIG. 2. Type 1(a) duct injury (arrow)

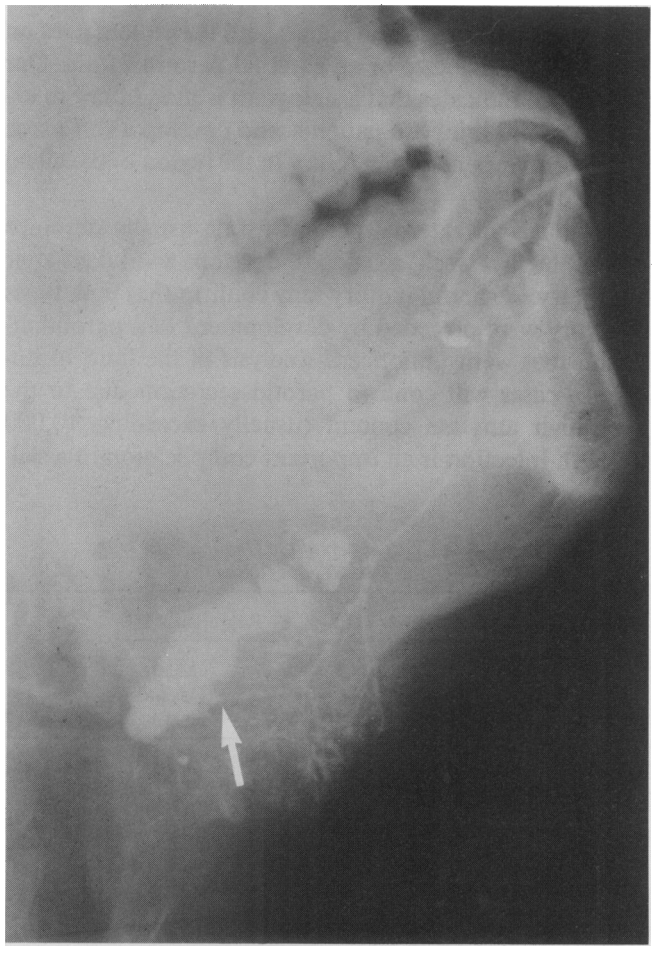

FIG. 1. Type 2 glandular injury. Arrow shows extravasation from a major intraparotid duct.

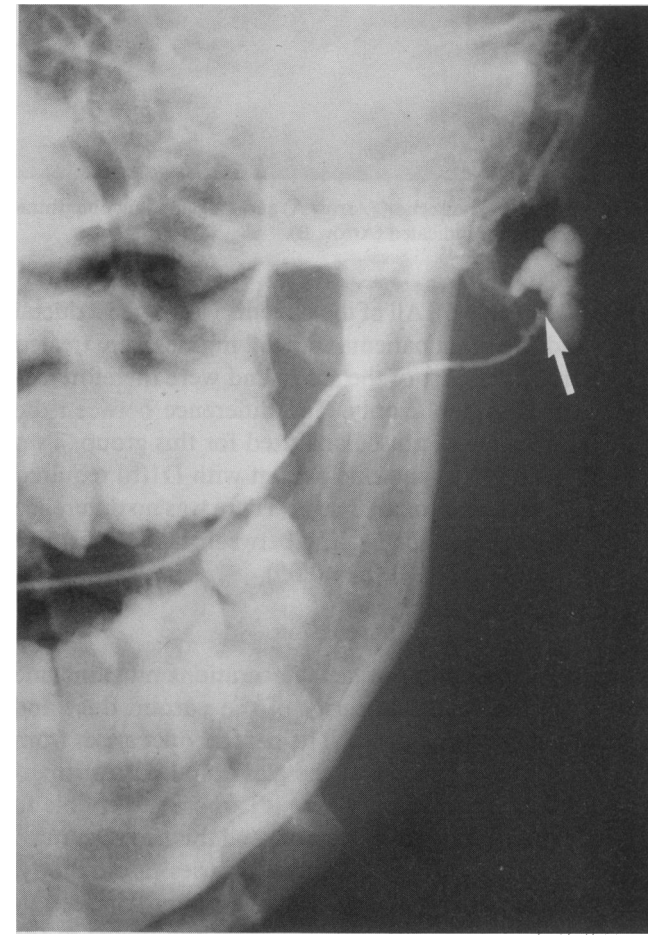

FIG. 3. Proximal duct transection (arrow) probably at duct-gland junction.

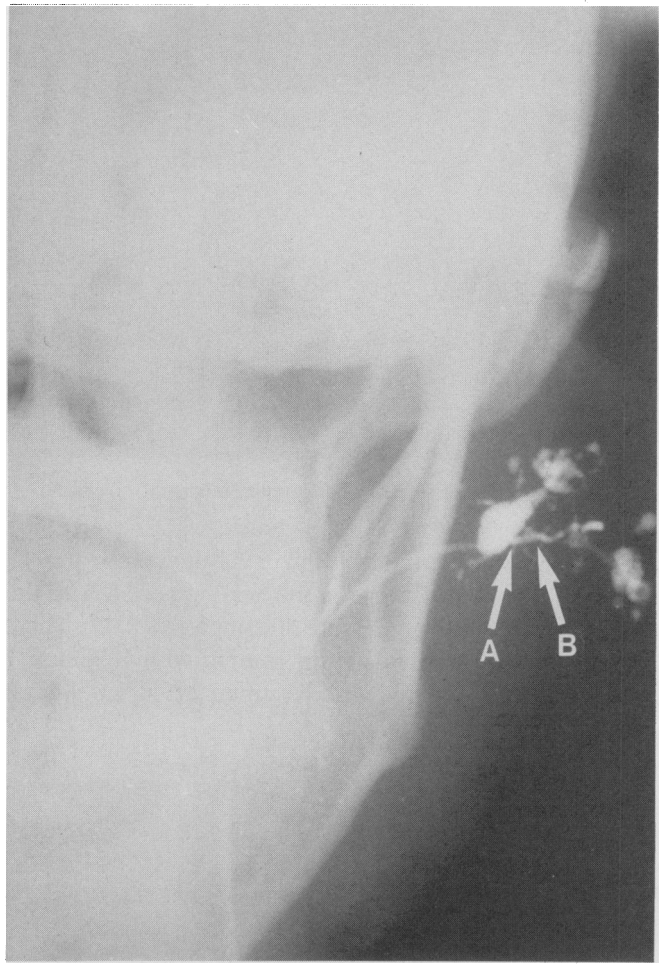

FIG. 4. Partial duct transection (Arrow A) at duct-gland junction. Intraparotid ductules are indicated (Arrow B).

($p < 0.001$)(Table 6). All of the patients with Type 2 ductal injury and five of six patients with G2 injuries were treated during the latter part of the study and were therefore entered into Regimen 2 only. The difference between the two regimens could not be analyzed for this group. Two patients with D2(b) and one patient with D1(b) required TPN for a mean period of 8 days. There was no difference in the mean period for healing between salivary fistulae and sialoceles (Table 3) ($p > 0.05$).

Discussion

A proper evaluation of facial lacerations must include demonstration of the integrity of the parotid duct and branches of the facial nerve. The parotid duct arises from the anterolateral portion of the gland and passes superficially over the masseter, where it is most susceptible to injury in penetrating facial trauma, and this site accounted for the majority of cases of ductal injuries in our study. The surface anatomy of the duct can be approximated by the middle third of a line drawn from the tragus to the midpoint of the upper lip. Any laceration crossing this line must be suspected of having damaged the parotid duct or its accompanying neurovascular bundle and should be meticulously assessed.[1]

TABLE 5. *Results of Conservative Management in the Different Types of Parotid Injuries*

Type	Total No.	Days To Healing
G1	9	6.3 ± 0.7
G2	6	10.3 ± 1.8
D1(a)	8	10.5 ± 2.2
D1(b)	20	21.7 ± 3.7
D2(a)	4	9.2 ± 1.4
D2(b)	3	20.0 ± 5.8

A parotid injury that is missed at the time of repair of the facial laceration usually presents as a parotid effusion after 24 hours.[7] Clinically the patient has limited or extensive soft tissue swelling of the face on the side of the injury. Unfortunately, parotid effusion is not a well-recognized entity and is often misdiagnosed as a soft tissue hematoma, as illustrated in our series. Diagnosis of a parotid injury at this stage may allow primary repair of the duct. If the parotid injury is missed at this stage, an inflammatory pseudocapsule limits further extravasation of saliva into the neck tissue planes, and the patient goes on to develop a sialocele or an external parotid fistula. Our experience indicates that a sialogram is mandatory to exclude parotid injury in patients who develop a soft tissue swelling after penetrating injury in the region of the gland or the duct.

Although an external parotid fistula usually develops within the first week, a sialocele develops 8–14 days after the injury. A careful inquiry may confirm that both types of injury were preceded by development of a parotid effusion that went unnoticed. Analysis of the fluid in uncertain cases will confirm parotid secretion due to the very high amylase content (usually exceeding 10,000 units/l). Infection is an important complication in a sial-

TABLE 6. *A Comparison of the Healing Rate with the Two Regimes Used*

	Regimen 1		Regimen 2		
Type of Injury	No.	Mean Days to Healing	No.	Mean Days to Healing	p-value*
G1	5	7†	4	4.5	0.13
G2	1	18	5	9	
D1(a)	2	20	6	7	0.04
D1(b)	10	34	10	9	0.0002
D2(a)	0		4	9	
D2(b)	0		3	20	
Total	18	24 ± 4	32	9.4 ± 0.9	0.001

* Regimen 1 *versus* Regimen 2.
† In this group only Pro-banthine and pressure bandages were used.

ocele and usually leads to an external salivary fistula, as demonstrated in a significant proportion of cases in our study.

The management of parotid fistulae and sialocele has been controversial[2–28]. The surgical techniques can be classified as those that divert parotid secretions into the mouth and those that depress parotid secretion either by ductal ligation or nerve sectioning. Conservative approaches include attempts to depress secretion by antisialogues or radiotherapy (Table 1). The method used in this study relies primarily on depressing parotid secretion (by administering nothing to the patient orally) so as to prevent meal-stimulated parotid secretion.

Techniques that have attempted to divert secretion into the mouth can be broadly divided into two groups (Table 1): 1) In the first group, attempts are made to reconstruct the duct to restore the passage for internal drainage of parotid secretion. 2) In the second group, a controlled internal fistula into the oral cavity is created that is held open by a polyethylene catheter into the proximal duct, wire, or seton around the fistula or T-tube or catheter drainage of the cavity of the sialocele into the mouth. The major problem with reconstructing the parotid duct has been the difficulty in identifying the proximal duct in the extensive scarring that forms around a sialocele with its associated significant risk of damage to the facial nerve.[26] The experience with many of the reconstructive procedures is limited (Table 2), and furthermore, the patency of the duct and that of parotid function in the long-term is not adequately documented. In recent years, the less extensive surgical procedures creating a controlled internal fistula have become popular (Table 2). However, the proximal duct probably does not remain patent with these procedures, as illustrated in a report from our institution where follow-up radioisotope scanning demonstrated progressive parotid atrophy in patients in whom a controlled internal fistula was created.[28]

Definitive studies on conservative methods that attempt to depress parotid secretion have not been previously reported, although occasional isolated case reports have claimed good results with the use of antisialogogues in glandular injury.[2] In this study, antisialogogues were adequate only in G1 injury and its use in other types of injury did not shorten the healing period. The effectiveness of tympanic neurectomy has varied in different reports, probably because of the variations in the pathway of Jacobsen's nerve.[14] Furthermore, this procedure is effective only in glandular injury because a high failure rate has been reported in ductal injuries (Table 2). The mean time reported for healing in glandular injuries after tympanic neurectomy is 9 days, which is similar to that of G2 injuries with Regimen 2 in the present study. However, a delay in healing of up to 6 months can occur after this procedure.[27] We believe that radiotherapy is not justified for benign disease where effective alternative methods of treatment are available, due to the significant association of secondary head and neck malignancies with its use. Furthermore, a significant failure rate has been reported with its use in ductal injuries (Table 2).

Extensive work has demonstrated that resting parotid secretion is negligible. In one study of 71 patients, resting parotid secretion was 0.03 ml/minute, with a range of 0–0.1 ml/minute.[31] In at least one third of the patients in this study, the resting secretion was 0 ml/minute. Studies in patients with parotid fistulae have shown that there is no secretion between meals and during sleep, whereas during a meal, large volumes of parotid secretion is produced.[32] The major stimuli for parotid secretion are gustatory and mechanical stimulation associated with mastication. Stimulated parotid secretion increases 30 times over basal secretion, averaging 0.9 ml/minute.[30] It is thought that stimulated parotid secretion is the major factor that delays or prevents healing of parotid fistulae.[14] The absence of reflex stimulation from mastication and chemical stimuli in patients administered nothing orally minimizes parotid secretion, and this probably allows the injured gland or duct to heal. An additional effect on the parotid gland produced by deprivation of oral intake comes from studies in rats indicating that prolonged starvation leads to disuse atrophy of the gland that may be mediated in part by the autonomic nervous system.[33] Whether a similar phenomenon occurs in humans who are fasted (as in this study) is not clarified. The importance of minimizing parotid secretion by administering nothing to the patient orally is illustrated by the marked difference in time taken to heal with Regimen 1 compared with the time taken with Regimen 2 (Table 6). Presumably, early feeding in Regimen 1 led to stimulation of the gland that retarded healing of the injury. This emphasizes the importance of minimizing parotid secretion during conservative therapy until the injury has healed.

Dynamic radioactive technesium (Tc-99m) scanning of the parotid gland has been shown to be a useful indicator of functioning parotid mass.[34] The effectiveness of the present conservative regimen was illustrated by serial dynamic parotid scanning in some of the patients. During the phase of conservative management (nothing administered orally), there was bilateral depression in parotid function probably due to minimal parotid secretion in the absence of reflex stimulation (Fig. 5). The healing of a complete ductal transection probably follows cicatrization and obstruction of the proximal duct lumen during conservative therapy. By contrast, we have found on follow-up sialography in Type 1(a) and 2(a) partial duct injury that, with healing, the duct may remain patent or occlude. The prognosis for duct patency in this instance probably depends on the severity of injury and the associated inflammatory response. As the injury healed, the

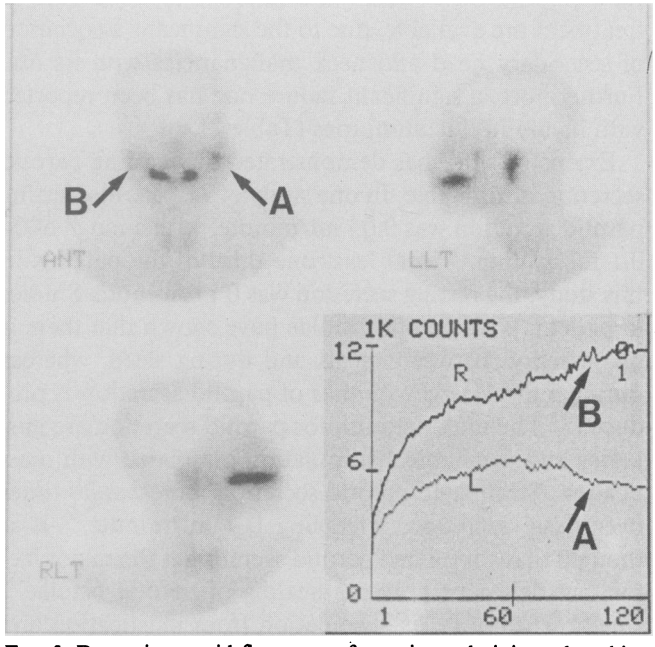

FIG. 5. Dynamic parotid flow scan of a patient administered nothing orally for 4 days. Arrows (A and B) demonstrate marked bilateral depression of parotid function. There is some retention of the isotope on the injured side (Arrow A).

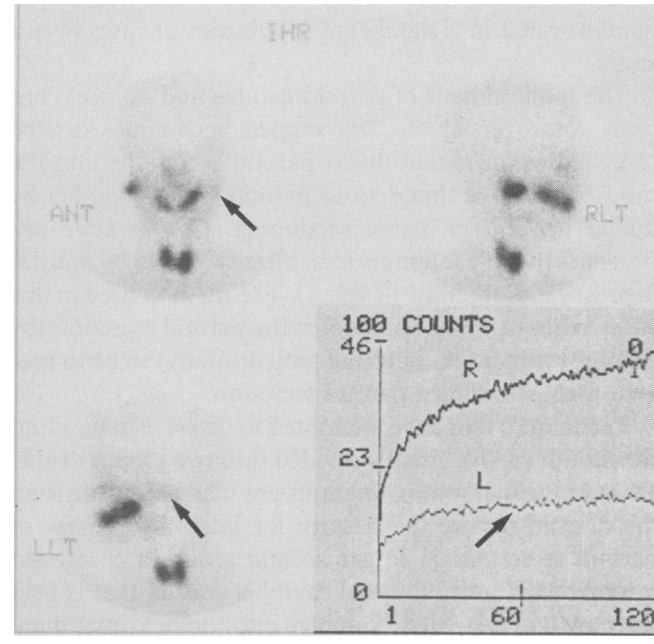

FIG. 7. Dynamic parotid flow scan 2 months after conservative treatment for D1(b) duct injury. Arrow shows the decrease in parotid function on the left due to progressive atrophy.

scans of patients with glandular injury and those with Type 1(a) and 2(a) ductal injury whose duct was patent reverted back to normal (Fig. 6), whereas in Types 1(b) and 2(b) ductal injury, there was progressive diminution in function over a period of months, probably due to glandular atrophy secondary to ductal obstruction from cicatrization (Fig. 7). This suggests that the prognosis for normal glandular function is excellent where duct patency is maintained after parotid injury; however, in those patients where healing leads to total obstruction of the proximal parotid duct, there is progressive atrophy of the parotid gland. This latter finding is consistent with previous experimental[35] and clinical studies[36] that have demonstrated that obstruction of the parotid duct after ligation leads to progressive parotid atrophy.

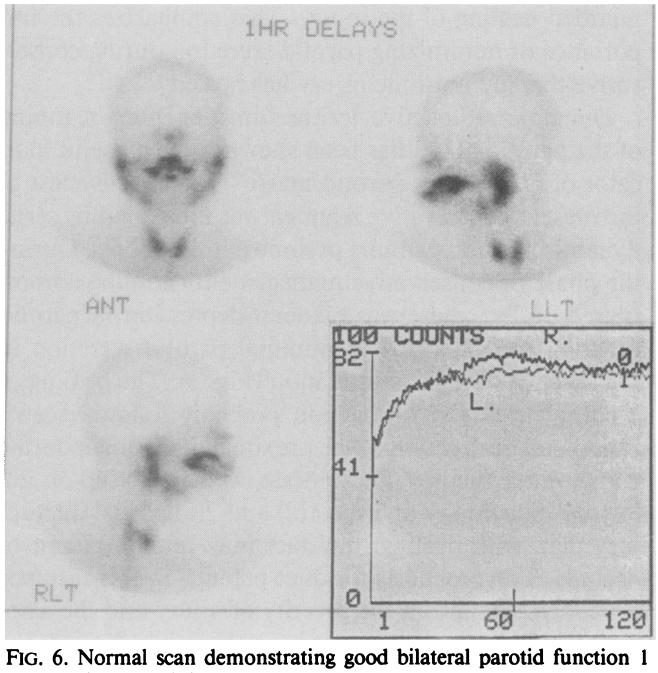

FIG. 6. Normal scan demonstrating good bilateral parotid function 1 month after a G2 injury.

The ability of the resting parotid gland to secrete against positive pressure (*e.g.*, after duct obstruction) seems to decline if this pressure is prolonged indefinitely.[32] This probably occurs because as the lobules of the gland are contained in relatively inelastic capsules, a sustained rise in duct pressure leads to compression of capillary and veins in the lobules, resulting in a reduction in the blood flow and diminution of secretion.[32] This sequence of events probably leads to the atrophy of the gland in the long-term. The findings of this study with sequential radioisotope scans support this sequence of events.

The healing of parotid fistulae and sialocele in patients with complete duct transection after both conservative therapy as described in this report and that after surgical internal diversion[28] probably result from glandular atrophy after ductal obstruction from cicatrization (Fig. 7). A similar process takes place in patients with duct ligation.[35,36]

The excellent results with conservative therapy found in this report, together with the risk to the facial nerve associated with surgical internal diversion and duct ligation procedures, warrants a conservative approach to parotid sialoceles and fistulae, particularly because the pro-

cesses governing healing are similar. Reconstructive procedures and parotidectomy should be abandoned because of the significant risk of morbidity to facial nerve,[26] and radiotherapy and tympanic neurectomy should be abandoned because of the inferior results and the associated risks of long-term morbidity with radiotherapy.

Ananthakrishnan and Prakash[26] have suggested that, compared with a sialocele, a fistulae is more resistant to cure. In this study, there was no difference ($p > 0.05$) between an external fistula and a sialocele in the time it took to heal (Table 3). The major prognostic factor for healing was the type of injury, based on sialographic findings (Table 5). G2 and Type 2 ductal injuries have not been identified as separate entities previously. Although G1 injury is probably insignificant in most cases, G2 injury is an important entity that carries the same prognosis as partial duct injury and probably accounts for most of the persistent glandular injuries reported in the literature. Disruption of duct-glandular junction [D2(b)] carries the worst prognosis and requires a prolonged period of conservative therapy including TPN. Surgical internal diversion may be more appropriate therapy in this group and requires further evaluation. In the patients of this study, the underlying ductal system and gland were normal. Whether this form of therapy will be as effective in fistulae with underlying parotid disease requires investigation.

Conclusions

The principles of management of the acute parotid injury is the accurate repair of all injured structures to return parotid function to normal, whereas in chronic parotid injury, the objective is to depress parotid secretion to allow natural healing processes to seal the injury. Preservation of parotid function here is not the primary goal because it probably will not be possible in complete ductal transections. In glandular and partial duct injuries, the prognosis for good function after conservative treatment is excellent. Limited conservative therapy with Pro-banthine and aspiration only may be sufficient in G1 injury; however, G2 and ductal injury patients should be administered nothing orally until the injury has healed. Based on this experience, we believe that the role of surgery is limited and should be used only occasionally where a sialocele or fistula persists after an adequate period of conservative therapy.

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